



Tissue Oxygen Utilization (Update in Intensive Care and Emergency Medicine)

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Disturbances in peripheral O extraction can be produced in dogs treated with 2 endotoxin and thereby provide an opportunity to test theories for the origin of pathological O supply dependency or to try different treatment modalities. The 2 most serious deficiency in the current animal models is the inability to mimic the increased O demand that is observed in patients at O₂ delivery rates in excess of 2 normal. A particular feature of this increased O demand is that it apparently does 2 not stimulate increased O₂ extraction, although the limitation in O extraction has 2 not been explored in patients by lowering O₂ supply, for obvious reasons. At least two possibilities to account for increased O₂ demand could be investigated in animal models, however. The amount of O₂ that is utilized in extramitochondrial pathways, which is normally on the order of 10%, may be greatly increased in ARDS and sepsis by O radical formation. There is presently no information 2 concerning how much O₂ might be used in this way. Another strong possibility is that mitochondrial injury, perhaps as a result of O₂ radical formation, uncouples oxidative phosphorylation. Some evidence presently in the literature supports this idea [19]. Indeed, the association of increased blood lactate levels with higher than expected O₂ demands makes uncoupling a very attractive hypothesis that warrants further investigation in animal models using such agents as 2,4-dinitrophenol. References 1.

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